METHODS The levels of serum CRP and blood Hcy in 280 patients suffered from coronary heart disease (including stable angina pectoris, unstable angina pectoris and acute myocardial infarction) and 80 healthy people from January 2013 to December 2014 were detected. The data were analyzed by SPSS 21.0.

RESULTS The levels of serum CRP was 13.05±3.68mg/L and blood Hcy was 21.17±8.63 μmol/L in patients with coronary heart disease were higher than those in the control group(5.02±1.60 g/L, 8.15±2.03 μmol/L), the differences were statistically significant($P < 0.05$). The levels of serum CRP and blood Hcy in the acute myocardial infarction subgroup of coronary heart disease patients(19.62±3.03 mg/L, 32.10±7.02 μmol/L) were higher than those in the unstable angina pectoris subgroup(12.98±6.11 mg/L, 20.82±6.04 μmol/L)($P < 0.05$); while, the levels of serum CRP and blood Hcy in the unstable pectoris angina subgroup were higher than those in the stable angina pectoris subgroup[(7.65±4.81) mg/L,(14.01±4.30) μmol/L], the differences were statistically significant ($P < 0.05$).

CONCLUSIONS The levels of serum CRP and blood Hcy are high expression in patients with coronary heart disease, and their expression level can be used as predictors to prompt the severity and type of coronary heart disease.

GW26-e2480

Long-term follow-up study of peripheral blood EMPs, EPCs levels in acute coronary syndrome patients with or without diabetes

Yashu Kuang,[1] Ying Li[1]
[1]Department of Cardiology, Shanghai East Hospital, Medical school, Tongji University

OBJECTIVES Explore the levels of peripheral blood $CD144^+$ / Annexin V^+ EMPs, EPCs in patients of acute coronary syndrome (ACS) with or without diabetes during long-term follow-up. Disscuss the relationship among EMPs, EPCs, coronary artery acute ischemic events and Abnormal glucose metabolism.

METHODS Study included 66 patients with acute coronary syndrome (ACS) according to the coronary angiography. *Based on clinical symptoms and cardiac marker levels, the patients were divided into two groups, A group:*diabetes mellitus, acute coronary syndrome (ACS) *(n=45), B group:* acute coronary syndrome (ACS) *(n=21).* Two groups of patients were given drug treatment and atorvastatin and followed up for 6 months. Respectively, test the levels of $CD144^+$ Annexin V^+ EMPs, EPCs in peripheral blood and clinical indexes, the presence of ischemic symptoms before and after the follow-up.

RESULTS During the 6 months follow-up, all of the patients have no abnormally elevated myocardial markers, sudden death due to coronary heart disease (CHD) and again revascularization readmission to the hospital clinical events. The level of $CD144^+$ / Annexin V^+ EMPs has no significant difference before and after follow-up between the two groups. However, no matter A or B, the level of $CD144^+$ / Annexin V^+ EMPs before follow-up was significantly higher than after follow-up ($P < 0.05$), and before and after follow-up there is no correlation between glycosylated hemoglobin and EMPs in group A. The level of EPCs of group A was significantly higher than that of group B ($P < 0.05$), and it was positively correlated to glycosylated hemoglobin ($r=0.457$). After follow-up, The level of EPCs of group A was significantly lower than that of group B ($P < 0.05$), and it was negative correlated to glycosylated hemoglobin ($P < 0.05$, $r = -0.365$). However, no matter A or B, the level of EPCs before follow-up was significantly higher than after follow-up ($P < 0.05$).

CONCLUSIONS No matter combining diabetes mellitus or not, acute ischemic events caused endothelial dysfunction is the dominant factor of peripheral blood EMPs levels, also the main cause of activating repair mechanisms to promote EPC mobilization. Diabetes patients in acute coronary events release more EPCs, which are positively related with glycosylated hemoglobin levels. But when atherosclerotic plaque turns to stable, the ability of repairing is much lower in patients with diabetes, and has negative correlation with glycosylated hemoglobin levels

GW26-e3574

Adiponectin stabilizes aortic plaques in ApoE-/- mice via regulating the level of autophagy

Yanwei Yu, Jingjie Li
First affiliated hospital of Harbin medical university

OBJECTIVES Adiponectin (APN), an adipose-derived adipokine, offers anti-atherogenic effects although the precise mechanism remains unclear. Autophagy reported as the major intracellular degradation system can be induced in stress conditions, such as atherosclerosis and oxidative stress. This study was designed to examine the correlation between APN and autophagy in apolipoprotein E-deficient (ApoE-/-) mice.

METHODS Adult ApoE-/- mice were fed a high fat diet for 12 weeks. After 8 week feeding, mice were treated with 10 ug/kg APN or vehicle every day for 4 weeks. The size of aortic plaque was measured by oil red O staining and autophagosomes were detected by transmission electron microscope. Western blot was used to evaluate the expression of autophagy maker protein, LC3II.

RESULTS The size of aortic plaque was reduced by APN. And the levels of autophagosomes decreased in APN group compared with control group. Furthermore, APN also decreased LC3II and LC3II/I protein expression ratio.

CONCLUSIONS These data suggest the autophagy in ApoE-/- mice caused by high diet could be ameliorated by APN.

GW26-e0455

Establishing the renalase gene low-expression modal in cardiac tissue of Sprague-Dawley rats via lentivirus-mediated RNA interference technology

Xiaogang LI, Weihong Jiang
The Third Xiangya Hospital of Central South University

OBJECTIVES Renalase is a novel secretory amino oxidase and expressed in kidney and heart. To research the protective mechanism of renalase in local heart tissue, we established the low-expression renalase model with lentivirus-mediated RNA interference technology.

METHODS Three renalase-targeting oligonucleotides were designed after analyzing the mRNA of renalase. Lentivirus particles were prepared by LV expression Systems (using the trono 3 plasmid component system), and then, LV-RNLS-shRNAs and LV-NC-shRNA were respectively transfected into H9C2 cells in different culture dishes. The optimal oligonucleotide was screened by real-time PCR and western blotting. The renalase gene low-expression in the heart tissue of rats via pericardial cavity injection. And real-time PCR and western blotting were used to detect renalase gene expression in the heart.

RESULTS In the cell screening experiment, the efficacy of the inhibition of renalase mRNA expression was 93.7%, and that of renalase protein expression was 83.1% in H9C2 cells. When the oligonucleotide was injected into the pericardial cavities of the SD rats on the 10th day, it inhibited 63.9% of the expression of renalase protein in the heart tissues.

CONCLUSIONS LV-Rnls-RNAi (19813-1) can be used to establish an optimal renalase low-expression model for the subsequent experiments.

OTHERS

OTHERS

GW26-e2302

Non-vitamin K Antagonist Oral Anticoagulants (NOACs) in Patients with Atrial Fibrillation and Heart Failure: A Systemic Review and Meta-analysis of randomized trials

Qinmei Xiong,[1,2] Yeecheng Lau,[2] Keitaro Senoo,[2] Kui Hong,[1] Gregory YH Lip[2]
[1]Cardiovascular department, the Second Affiliated Hospital of Nanchang University, Nanchang, China; [2]University of Birmingham Centre for Cardiovascular Sciences, City Hospital, Birmingham, United Kingdom

OBJECTIVES The relative efficacy and safety of non-vitamin K antagonist oral anticoagulants(NOACs) against warfarin have been assessed for stroke prevention in atrial fibrillation(AF) in several clinical subgroups. However, no pooled analysis has been undertaken across the four landmark phase 3 randomized controlled trials (RCTs) to assess the effects of all NOACs against warfarin in the subgroup of patients with AF and heart failure (HF). We performed a systemic review and meta-analysis of RCTs to determine the relative efficacy and safety of NOACs against warfarin among subgroup patients with AF and HF. Additionally, we compared outcomes between AF patients with HF and without HF.